

- 5. (Amended) The method of claim 4, wherein the inhibitor decreases expression of a gene encoding ERK1/2, a MEK and/or a JNK.
- 6. (Amended) The method of claim 5, wherein the inhibitor interacts with an ERK1/2, a MEK and/or JNK gene.
- 8. (Amended) The method of claim 3, wherein the inhibitor decreases the activity of ERK1/2, a MEK and/or a JNK.
- 9. (Amended) The method of claim 6, wherein the inhibitor interacts with ERK1/2, a MEK and/or a JNK protein.
- 10. (Amended) The method of claim 6, wherein the inhibitor inhibits ERK1/2, a MEK and/or JNK phosphorylation.

Please insert new claims 19-28:

- 19. (New) The method of claim 1, wherein the inhibitor is a dominant negative mutant of ERK1/2, a MEK and/or a JNK.
- 20. (New) The method of claim 1, wherein the subject is overweight or obese.
- 21. (New) The method of claim 1, wherein the disease or condition is caused, or contributed to, by TNF-α induced lipolysis.
- 22. (New) The method of claim 1, wherein the disease or condition is caused, or contributed to, by basal lipolysis.
- 23. (New) The method of claim 22, wherein the inhibitor does not interact with a PPAR-y receptor and the inhibitor is not sodium salicylate.
- 24. (New) The method of claim 23, wherein the inhibitor is selected from the group consisting of an antisense molecule, a triplex molecule, a ribozyme and a dominant negative mutant targeted to ERK1/2 or a MEK.
- 25. (New) The method of claim 1, further comprising determining the level of activity of ERK1/2 or a MEK in the subject.
- 26. (New) The method of claim 25, wherein the level of activity of ERK1/2 or a MEK is determined in a sample of fat cells from the subject.

- 27. (New) The method of claim 1, wherein the inhibitor is administered in the presence of a carrier that facilitates entry of the inhibitor into cells of the subject.
- 28. (New) The method of claim 1, wherein the inhibitor is administered locally.